DISSERTATION ON

A Study on Vertigo – Evaluation of Bedside Tests and Aetiopathology

Submitted in partial fulfilment of

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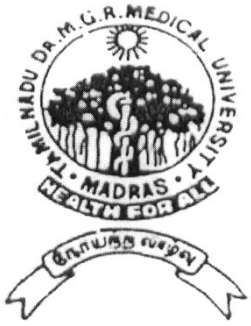

MADRAS MEDICAL COLLEGE

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CERTIFICATE

This is to certify that this dissertation entitled **"A study on Vertigo – Evaluation of Bedside Tests and Aetiopathology"** submitted by **Dr.**P.Muthukumar appearing for D.M., Degree examination in August 2007 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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DECLARATION

I solemnly declare that the dissertation titled "**A study on Vertigo – Evaluation of Bedside Tests and Aetiopathology"** is done by me at Institute Of Neurology, Madras Medical College & Govt. General Hospital, Chennai, during 2004-2007 under the guidance and supervision of Prof. V.Natarajan,

The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of requirements for the award of D.M., degree in Neurology.

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I should thank each patient for the whole-hearted cooperation for this study.

CONTENTS

INTRODUCTION

INTRODUCTION

Vertigo is defined as the 'hallucination' of movement, either of self (subjective) or the environment (objective) (1).

Usually the patient uses various terms (eg.) Bouncing, Oscillating, Staggering, Swimming, Twisting Rolling, Spinning, Rocking, Lightheadness, Imbalance, Floating, Fainting, Falling (1).

For the most part they are benign but always there is the possibility that they signal the presence of an important neurological disorder (2).

Diagnosis of the underlying disease demands that the complaint of vertigo be analyzed correctly, the nature of the disturbance of function being determined first and then its anatomic localization (2).

A careful history and physical examination usually affords the basis for separating true vertigo from the dizziness of the anxious patient and from the other types of pseudo vertigo (2)

This study evaluates the vertigo patients based on history, examination and relevant investigations to establish the etiology for the vertigo.

Objective of the Study

OBJECTIVE OF THE STUDY

- To evaluate the bedside tests used in vertigo
- Find out the cause for vertigo by using history, bed side examination and investigations.

MATERIAL AND METHODS

MATERIAL AND METHODS

Study design – Prospective study

Study place – Govt. General Hospital the teaching hospital of Madras Medical College.

Study period – October 2004 to April 2007

Inclusion Criteria – Patients attend the neurology OPD with chief complaint of Vertigo.

Exclusion Criteria – Known case of head injury with vertigo
Known cardiac patients with vertigo
Known case of seizures with vertigo

Total No of Patients - 110

Maneuver:

All the patients were subjected to a thorough history, clinical examination and various bed side tests like Head thrust test, Dynamic visual acuity, Head shaking test, Dix Hall pick's test, Fukuda's test and Calorie test and investigations like CT / MRI brain, X-Ray neck, Blood sugar, Lipid profile, Carotid vertebral Doppler.

The Head Thrust test: Method: the patients's head is held firmly on each side and the patient is asked to fixate on any one point – say the examiner's nose. The head is moved slowly from side to side to see if the eyes are remaining fixed on one point (the examiner's nose). Once the examiner is satisfied that the patient is following the instructions, the head is rapidly brought back into the midline. If the VOR is normal, the

eyes do not move at all. If there is vestibular imbalance, the VOR on the affected side is hypoactive and the eyes move with the head. When the head is brought back to the midline, there is a re-fixation saccade. The examiner carefully looks for this re-fixation movement of the eyes. This saccadic re-fixation only occurs after rotation of the head to the affected side and persists for a very long time. The Head Thrust test is very reliable for unilateral vestibular hypo function (3).

Dynamic Visual Acuity: Method: Ask the patient to read the smallest line possible on a Snellen's eye chart with best corrected vision. Repeat the visual acuity (VA) while passively shaking the patient's head at 2Hz. Record the number of lines "lost" during the head shake. If the VOR is normal, the eyes remain fixed on the target line and the visual acuity does not change. If the VOR is hypoactive, the eyes move with the head and are no longer fixed on the target line, resulting in visual degradation. Loss of three or more lines from static VA indicates vestibular dysfunction and is a good test for vestibular hypofunction due to ototoxicity or age (3).

The Head Shaking test: Method: The patients head is pitched down 30' and oscillated at 2 Hz for 20 seconds. An abnormal test is elicitation of jerk nystagmus. Post head shake nystagmus is considered pathologic of vestibular imbalance. In most cases a peripheral is identifies with the fast phase beating towards the unaffected (stronger) ear (3).

The Dick-Hallpike test is a manoeuvre which is specifically positive if a patient has

benign paroxysmal positional vertigo (BPPV). It is intended to stimulate the vertical SCC. The anterior and contralateral posterior SCCs are approximately parallel in a plane orientated 45’ from the sagittal plane. With the head turned 45’ to one side, moving patient from sitting to supine results in rotation in the plane of the canal pair, ie. In the horizontal plane. In the normal patient, nystagmus occurs during the manoeuvre but not after it. In BPPV, a change of position causes movement of the endolmph elicitin g vertigo. The nystagmus starts at a latency of 4-10 secs and lasts upto 30secs. Involvement of the posterior SCC results in up-beating nystagmus with a rotary component, the upper pole of the eye beating towards the lower ear. BPPV results from debris moving freely in the vertical SCCs, usually the posterior (3).

Fukuda test (Stepping Test of Underberger)

The patient to march in place with eyes closed and arms outstretched normally less than 15 Degrees or so of rotation is displayed Asymmetry of labyrinthine function is manifest as excessive rotation away from the diseased side (4).

Calorie test

The patients head ideally tilted forward 30 degrees from the horizontal, each auditory canal is irrigated for 30 seconds, first with water of 30˚ C, and then at 44˚ C with a pause of at least 5 minutes between each irrigation. In normal persons cold water induces a slight tonic deviation of the eyes to the side being irrigated, followed after a

latent period of 20 seconds by nystagmus to the opposite side (direction of fast phase). Warm water induces nystagmus to the irrigated side (2).

Caloric testing will reliably answer whether the vestibular end organs react and comparison of the responses from the two ears will give which one is paretic (2)

Nystagmus

The presence of spontaneous or induced nystagmus is of crucial importance in making a diagnosis of peripheral or central causes of imbalance (3).
Spontaneous nystagmus of a peripheral origin is usually due to lesions either of the labyrinth or eighth cranial nerve. The characteristics of nystagmus of a peripheral origin are as follows: (3)

a) Mixed – horizontal plus rotational / tortional.
b) Presence of fixation suppression – if there is a gaze evoked nystamus while the patient is staring at a blank wall, asking the patient to fixate on your finger, suppresses the nystagmus i.e. the nystagmus is so-to-say “fatiguable”
c) The nystagmus is intense, i.e. it intensifies (increases in amplitude) in the direction of the fast phase,
d) It is direction fixed i.e. it does not change direction with gaze. Usually in an irritative lesion, it is in the direction of the affected ear and beats towards the

unaffected ear if the lesion is destructive.

Spontaneous nystagmus of central origin is usually due to lesions of the brainstem, cerebellum or rarely certain areas of the cerebrum. The characteristics of nystagmus of central origin are quite the opposite of peripheral nystagmus (3).

Thus the characteristics of central nystagmus are:

a) pure – purely horizontal, vertical or torsional,
b) absence of fixation-suppression – it is “non-fatiguable” when one fixates over the examiner’s finger,
c) less intense – does not intensify in amplitude in the direction of fast gaze,
d) direction changing with gaze - left beating nystagmus with left gaze.

Nystagmus is dampened by convergence, therefore, do not hold the finger very close to the eyes – hold it at least 14 inches away. Primary position nystagmus is suppressed by fixation. The maneuvers to suppress fixation are to ask the patient to stare at a totally blank wall, the hand held ophthalmoscope method (3).

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Spatial orientation is largely automatic but complex. Continued sensory monitoring assesses the position of the body in space, in relation to the surrounding environment. The 5 sensory modalities constantly sample position and motion: vision, vestibular sensation, proprioception, touch and pressure, and hearing. Normally the brain integrates the input from each of these sensory modalities giving a comprehensive image of position and motion in space. This process enables us to maintain balance, move about, and interact with other objects. When the orienting image is unreliable, we become uncertain of position and the result is a sensation of spinning or vertigo. When a patient presents with this type of dizziness, the clinician must next determine whether the symptom is central (brain) or peripheral (inner ear, 8^{th} cranial nerve) in origin.

Peripheral causes of vertigo

1. Peripheral vestibulopathy (includes labyrinthitis, vestibular neuronitis, and acute and recurrent peripheral vestibulopathy)
2. Benign positional vertigo (includes benign positional nystagmus, benign paroxysmal vertigo)

3. Post-traumatic vertigo
4. Vestibulotoxic, drug-induced vertigo
5. Meniere's disease

6. Other focal peripheral diseases (includes local bacterial infection, degeneration of hair cells, genetic anomalies of labyrinth, cupulolithiasis, tumor of eighth nerve, otosclerosis, fistula of labyrinth, and, rarely, focal ischemia) (1)

Central neurological causes of vertigo

1. Brainstem ischemia and infarction
2. Demyelinating disease: multiple sclerosis, post infectious demyelination, remote effect of carcinoma
3. Cerebellopontine angle tumor: acoustic neuroma, meningimo, chlesteatoma, met static tumor etc.
4. Cranial neuropath: focal involvement of eighth nerve or in association with systemic disorders
5. Intrinsic brainstem lesions (tumor, arteriovenous malformation)
6. Other posterior fossa lesions (primarily other intrinsic or extra – axial masses of the posterior fossa, such as hematoma, metastatic tumor, and cerebellar infarction)
7. Seizure disorders (rare)
8. Heredofamilial disorders (such as spinocerebellar degeneration) (1)

Systemic causes of vertigo

1. Drugs (including anticonvulsants, hypnotics, antihypertensive, alcohol, analgesics, tranquilizers)
2. Hypotension, presyncope (including primary cardiac causes and postural hypotension from a wide variety of causes)
3. Infectious diseases (including syphilis, viral and other bacterial meningitides, and systemic infection)
4. Endocrine diseases (including diabetes and hypothyroidism)
5. Vasculitis (including collagen vascular disease, giant cell arteritis, and drug – induced vasculitis)
6. other systemic conditions (including hematological disorders [polycythemia, and dysproteinemia], sarcoidosis granulomatous diseases and systemic toxins) (1)

Characteristics of peripheral versus central positional vertigo (1)

Symptom or sign	Peripheral	Central
Latency (time to onset vertigo Or nystagmus)	0-40 sec (mean 7.8*)	No latency; begins immediately
Duration	<1 min	Symptoms may Persist (signs and Symptoms of single Episode)
Fatigability (habituation)	Yes	No

(Lessening signs and symptoms With repetition of provocative Maneuver)		
Nystagmus direction	Direction fixed, torsional,Up, upper pole of eyes toward ground	Direction changing, variable
Intensity of signs and Symptoms	severe vertigo, marked nystagmus, nausea	usually mild vertigo, less intense Nystagmus, rare Nausea
Reproducibility	Inconsistent	More consistent

Peripheral Vestibulopathy 12

Peripheral vestibulopathy has been described as *vestibular neuronitis, labyrithitis, or viral neurolabyrinthitis.* Such terms imply an inflammatory mechanism, which is unproved. Vestibular neuronitis, strictly speaking, is characterized by single or recurrent sudden episodes of true vertigo lasting from hours to days and often associated initially with vomiting. When the condition is associated with hearing loss, the entire labyrinth is assumed to be involved, and the term labyrinthitis is used. Despite this technical distinction, many neuro-otologists, otologists, and neurologists use the terms vestibular neuronitis and labyrinthitis interchangeably, whether or not

auditory symptoms are present. In such patients, the vertiginous sensation may be provoked by head movement but not necessarily by a particular head position (1).

Whether isolated viral involvement of the vestibular nerves is a cause of acute or episodic vertigo is controversial. Many prefer the term acute or recurrent peripheral vestibulopathy. In the acute phase, most patients present with sudden severe vertigo, nausea, and vomiting without any hearing disturbance or facial weakness. The acute symptoms usually resolve in a few days to a week but may recur in weeks or months. If true vertigo is part of the symptom complex, the condition is most likely to be associated with some disorders of the peripheral end organ. However, patients with either acute peripheral vestibulopathy or more commonly recurrent attacks may experience only a sensation of lightheadedness or floating or a feeling of “walking on tennis balls”. Even if the patient has had hundreds of episodes, it is important to try to determine whether any of them were associated with spinning vertigo. With time, the nature of the patient’s symptom complex may change, even with peripheral vestibulopathy, from vertiginous sensations to those of pure unsteadiness or disequilibration (1).

Epidemic and seasonal outbreaks of acute vertigo have suggested an infectious origin caused by viral disease, but this remains largely unproved. Viral labyrinthitis can also be part of a systemic viral infection, such as mumps, measles, infectious mononucleosis, or upper respiratory tract viral infections. Isolated viral infections of the labyrinth are also believed to cause the sudden onset of hearing loss, vertigo, or both,

in children and adults, otitic herpes zoster's is an infection characterized by pain in the ear, followed in 1-10 days by a vesicular eruption in the external ear, when the seventh and eighth nerves are affected, there is a combination of facial weakness, hearing loss, and vertigo known as the Ramsay Hunt syndrome. Whenever vertigo is associated with severe car pain or facial pain, one must consider this possibility. A dysesthetic area of skin may precede, by many days, the appearance of the skin eruption (1).

Benign Paroxysmal Positional Vertigo[14]

Benign paroxysmal positional (or "positioning") vertigo (BPPV) is a symptom complex suggesting benign peripheral (end-organ) disease. It is a major cause of vertigo. Historical factors that should lead to the consideration of BPPV include the following: (1) symptoms associated with certain head positions, (2) episodic rotational vertigo of brief duration, (3) antecedent episode of severe rotary vertigo with or without nausea and vomiting, associated with an upper respiratory tract infection that suggests prior viral neurolabyrinthitis, (4) history of head trauma before attacks of vertigo, (5) most severe symptomatology early in the day, with lessening symptoms as the day progress, and (6) relative absence of spontaneous symptoms without head movement or position change. These symptoms, differentiated from central neurological symptoms, are outlined. The signs and symptoms of benign positional vertigo are

transient and rarely last longer than 40 seconds. They usually occur when a certain position is assumed, such as lying down or turning in bed. Depending on whether the symptom (vertigo) or sign (nystagmus) is being emphasized, this condition can be called benign paroxysmal positional nystagmus or BPPV. Physical examination findings include (1) vertical rotary benign positional paroxysmal nystagmus produced by provocative maneuvers. (2) Latency to onset of symptoms once precipitation head position is achieved, (3) short-duration nystagmus (3-30 seconds), and (4) adaptation of nystagmus and symptoms (i.e., disappearance with repeated maneuvers). The findings of the typical nystagmus on assumption of certain head positions are considered the most important physical finding in making the diagnosis of BPPV (1).

Diagnostic Criteria for Benign Paroxysmal Positional Vertigo

- Vertigo associated with a characteristic mixed torsional and vertical nystagmus provoked by the Dix-Hall pike test
- A latency (typically of 1 to 2 seconds) between the completion of the Dix-Hall pike test and the onset of vertigo and nystagmus
- Paroxysmal nature of the provoked vertigo and nystagmus (i.e., an increase and then a decline over a period of 10 to 20 seconds)
- Fatigability (i.e., a reduction in vertigo and nystagmus if the Dix-Hall pike test is repeated (5).

In benign paroxysmal positional vertigo, the nystagmus fast phase is horizontal-rotary directed toward the lower car. The nystagmus fast phase is upward toward the

forehead when gaze is directed to the upper car. With the eyes in the central orbital position, the nystagmus fast phase is vertical upward and rotary toward the lower car (1).

Meniere's disease

Meniere's disease is characterized by attacks of severe vertigo and vomiting, tinnitus, fluctuating hearing loss, ill-described aural sensations of fullness and pressure and spontaneous recovery in hours to days. Usually, the patient develops a sensation of fullness and pressure associated with decreased hearing and tinnitus in a single ear. This is followed by severe vertigo which reaches peak intensity within minutes and slowly subsides over hours, with a persistent sense of disequilibration for days after an acute episode. Occasionally severe attacks cause sudden falls to the ground. Consciousness is not lost in such episodes, although of the accompanying vertigo nausea. The most consistent pathological finding in Meniere's disease is an increase in the volume of the endolymphatic fluid and distension of the canals, hence the term endolymphatic hydrops (Saeed 1998). Although some specific causes, such as bacterial, viral, and syphilitic infections, may lead to the same pathological changes and symptoms, most cases are idiopathic (1).

Diagnostic Criteria for Meniere's disease

Meniere´s disease is defined as recurrent, spontaneous episodic vertigo, hearing loss, aural fullness and tinnitus. Recurrent endolymphatic hypertension (hydrops) is

believed to cause the episodes (6).

According to the guidelines from AAO-HNS Committee of Hearing and Equilibrium the three major symptoms are described as follows: (6)

Vertigo

- Recurrent, well-defined episodes of spinning or rotation
- Duration ranging from 20 min to 24 h
- Nystagmus associated with attacks
- Nausea and vomiting during vertigo spells common
- No neurologic symptoms with vertigo

Deafness

- Hearing deficits fluctuate
- Sensorineural hearing loss
- Hearing loss progressive, usually unilateral

Tinnitus

- Variable, often low pitched and louder during attacks
- Usually unilateral on the affected side
- Subjective

Possible Meniere's disease

Episodic vertigo of the Meniere's type without documented hearing loss, or

Sensorineural hearing loss, fluctuating or fixed, with dysequilibrium but without definitive episodes

Other causes excluded

Probable Meniere's disease

One definitive episode of vertigo

Audio metrically documented hearing loss on at least one occasion

Tinnitus or aural fullness in the treated ear

Other causes excluded

Definite Meniere's disease

Two or more definitive spontaneous episodes of vertigo 20 minutes or longer

Audio metrically documented hearing loss on at least one occasion

Tinnitus or aural fullness in the treated ear

Other cases excluded

Certain Meniere's disease

Definite Meniere's disease, plus histopathologic confirmation (6).

Vestibular neuronitis:

This term is based on their clinical findings which suggested that the syndrome is caused by an isolated lesion of the vestibular nerve and its central connections (7). Vestibular neuronitis is diagnosed using three clinical diagnostic criteria:

- Vertigo: usually sudden onset:
- An absence of cochlear symptoms or signs (deafness and tinnitus);
- An absence of associated neurological symptoms and signs.

A non-essential criterion is a reduced or absent response to the caloric test (7).

VERTIGO IN MIGRAINE

Vertigo has been found to occur significantly more frequently in patients with migraine than in controls (8,9,10) and a high prevalence of migraine has been found in vertigo sufferers (11, 12).

"Benign recurrent vertigo"(13, 14) of adults which may be considered as a condition like "***vertiginous aura without headache"*** is not included in IHC classification. However benign recurrent vertigo is considered by many researchers to be a migraine equivalent. If benign recurrent vertigo has certain associated migranious symptoms during some episodes, together with a history of typical migranious attacks outside the

episodes of vertigo, it can be classified as ***"Definite migranious vertigo"*** and ***"Probable vertiginous vertigo"*** respectively (15).

I BASILAR MIGRAINE

"Basilar migraine" consists of aura and headache.

The ***AURA*** should include at least two of the following: vertigo, tinnitus, decreased hearing, ataxia, dysarthria, double vision, hemi field visual symptoms (both eyes and both fields), bilateral paraesthesia, bilateral hemiparesis, decreased level of consciousness (12).

II DEFINITE MIGRAINOUS VERTIGO

- Episodic vestibular symptoms of at least moderate severity (rotational vertigo, other illusory self or object motion, positional vertigo, head motion intolerance).
- At least two of the following migraninous symptoms during at least two vertiginous attacks: (a) migraninous headache; (b) photophobia, (c) phonophobia; (d) visual or other aurae.
- Attacks of migraine (outside episodes of vertigo) according to HIS migraninous
- Some central and/or peripheral vestibular abnormalities may be found in

vertigo-free periods.

- Other causes ruled out by history, physical examination and other appropriate investigations (12).

III <u>PROBABLE MIGRAINOUS VERTIGO</u>

- Episodic vestibular symptoms of at least moderate severity (rotational vertigo, other illusory self or object motion, positional vertigo, head motion intolerance)
- At least one of the following, in relation to at least one vertiginous attack: (a) **migrainous** headache, (b) photophobia, (c) phonophobia, (d) migraine-specific triggers e.g. specific foods, sleep irregularities, hormone changes
- Response to migraine prophylactic drugs.
- Migraine (outside vertiginous attacks) according to the **criteria** of the IHS
- Some central and/or peripheral vestibular abnormalities may be found in vertigo-free periods.
- Other causes ruled out by history, physical examination and other appropriate investigations (12).

BENIGN RECURRENT VERTIGO

- Episodic vertigo, occasionally with tinnitus but without hearing loss
- May be accompanied by nausea/vomiting and ataxia
- Nystagmus may be observed during the episode
- Duration: minutes to hours, usually less than one hour , or hours to days
- Episodes of **migrainous** headaches outside vertiginous episodes, and/or positive family history of migraine.
- Normal audiometric findings, or no asymmetry if there is an incidental hearing loss
- Differential diagnosis: vestibular hydrops, other episodic vestibular disorders, epilepsy (12).

Pathophysiology: In 1992, Cutrer and Baloh developed the most commonly accepted theory regarding the pathophysiology of migraine-associated vertigo. These authors propose that episodes of dizziness of a duration similar to that of a migraine aura (<60 min) that are time-locked with the headache most likely have the same pathophysiologic mechanism (eg, spreading wave of depression) as other aura phenomena (16).

According to the spreading depression theory, some type of stimulus (eg, chemical, mechanical) results in a transient wave front that suppresses central neuronal activity. This depression spreads in all directions from its site of origin. Neuronal depression is

accompanied by large ion fluxes, including increases in extracellular K^+ and decreases in extracellular Ca^{++}. These changes result in a reduction in cerebral blood flow in the areas of spreading depression. However, most patients with migraine-associated vertigo have dizziness that occurs independent of the headache (16).

Cutrer and Baloh suggest that when dizziness is unrelated to headache, the dizziness occurs from the release of neuropeptides (ie, neuropeptide substance P, neurokinin A, calcitonin gene–related peptide [CGRP]). Neuropeptide release has an excitatory effect on the baseline firing rate of the sensory epithelium of the inner ear, as well as on the vestibular nuclei in the pons (16).

5-HT has direct effects on the firing rate of vestibular nucleus neurons. Both the serotonergic and the peptidergic pathways possibly play a role in the development of the short and prolonged periods of dizziness in migraine-associated vertigo. No single hypothesis explains the headache or dizziness process in migraine at this time (16).

Sex: The epidemiology of migraine-associated vertigo corresponds to that of migraine in general. Migraine is present in 18% of females and in 6% of males aged 12-80 years. Peak ages are 30-45 years (16).

No diagnostic tests exist for migraine-associated vertigo (16).

In migraine-associated vertigo, the patient reports a history of acute-onset vertigo that lasts minutes to a few hours or many hours to days. Vertigo may precede, follow, or

appear simultaneously with the headache (migraine without aura), and vestibular symptoms range from mild to severe. Vertigo may occur with the additional symptom of aura (ie, visual and somatosensory paresthesias) before a headache (ie, migraine with aura). Less commonly, vertigo is a symptom of basilar migraine (17).

VERTIGO IN CEREBROVASCULAR DISEASE

Several mechanisms can cause vertigo and dizziness in persons with cerebrovascular disease. Presyncopal lightheadedness occurs with diffuse cerebral ischemia from any etiology (eg, orthostatic hypotension, myocardial infarction). Postural imbalance or disequilibrium results from loss of any component of the balance pathway (ie, vestibular, somatosensory, central integration, motor output) caused by transient ischemia or infarction. Vertigo can occur with cerebrovascular disease that involves the vertebrobasilar circulation, which supplies the labyrinth, the lateral pontomedullary region that contains the vestibular nuclei, and the cerebellum (17).

In vertebrobasilar artery (VBA) insufficiency, vertigo is sudden in onset, lasts only minutes, is associated with nausea and vomiting, and is usually accompanied by a range of neurologic deficits (eg, extremity weakness, numbness, in coordination, and dysarthria; diplopia; field defects; tinnitus: hearing loss; loss of consciousness; drop attacks). Isolated vertigo without additional symptoms can be the presenting manifestation of vertebrobasilar ischemia (17).

A subgroup of patients with episodic, spontaneous, or positional vertigo experience poor autonomic regulation. These patients usually have a low (<100 mm Hg) systolic blood pressure and associated symptoms of palpitations, chronic fatigue, sleeping disorders, cold extremities, and fainting spells. They also usually have a history of mitral valve prolapse (17).

Symptoms and signs of posterior fossa cerebrovascular disease include vertigo, tinnitus, and ataxia. Symptoms may be transient, permanent, recurrent, or isolated. Stroke syndromes that involve the posterior circulation vary depending on the involved territory. Wallenberg syndrome (ie, lateral medullary syndrome) appears with vertigo, nausea, vomiting, imbalance, ipsilateral facial numbness and weakness, diplopia, dysphagia, and dysphonia. Infarction of the dorsolateral pontomedullary region results in labyrinthine injury (ie, severe vertigo, nausea, vomiting, hearing loss) in addition to the signs and symptoms of Wallenberg syndrome (17).

Cerebellar infarction also occurs with severe vertigo, nausea, vomiting, and ataxia. When cerebellar infarction occurs without any other associated neurologic or audio logic symptoms, the presentation may be attributed to viral neuronitis (VN). Such cases of pseudo-VN usually occur after infarction of the nodulus and uvula, territory supplied by the medial branch of the posterior inferior cerebellar artery (mPICA).

Cerebellar signs that affect the extremities can be minimal or absent in mPICA infarction of the vestibulocerebellum. Infarction of the territory supplied by the anterior inferior cerebellar artery (AICA) rarely presents as pseudo-VN, since cochlear

ischemia manifested as hearing loss and additional brainstem signs, such as facial palsy, are typically present (17).

Basilar artery syndrome results from infarction of the pons. Symptoms include vertigo, hearing loss, ataxia, ophthalmoplegia, blindness, and regional sensory losses (17).

Cerebellopontine angle (CPA) tumors typically result in disequilibrium or unsteadiness rather than a sensation of vertigo. However, sudden change in tumor size with hemorrhage or disruption of regional blood flow to the labyrinth may precipitate vertigo. Tumors in the CPA are most likely to be vestibular schwannomas (17).

Pseudo-VN can be reliably differentiated from true VN by using the head thrust test and caloric testing (17).

Central vertigo syndromes that result from acute vascular events most commonly result from a combination of hypertension and regional atherosclerosis (17).

OBSERVATION AND RESULTS

OBSERVATION AND RESULTS

Total No of Cases - 110

Peripheral Causes - 75

Central Causes - 21

Systemic Causes - 14

TA28

Age Distribution of the Study population

Age	No of Cases	Percentage
11 – 20	7	6.3
21 – 30	20	18.1
31 – 40	39	35.4
41 – 50	23	20.9
51 – 60	13	11.8
61 – 70	5	4.5
71 – 80	3	2.7

35.4% of the patients were in the age group of 31-40 - Peak Incidence

2.7% of the patients were in the age group of 71-80 - Low Incidence

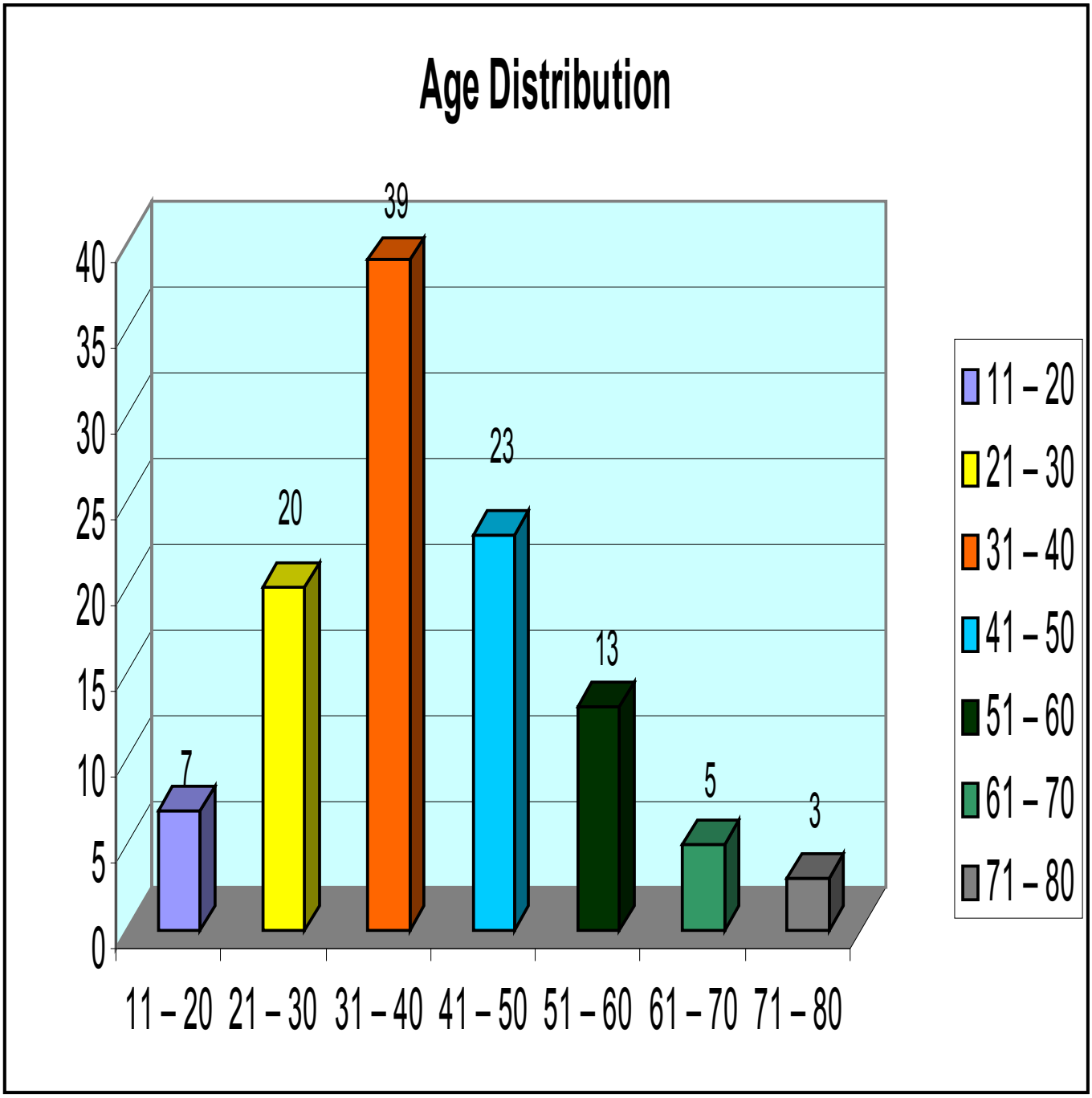

Age Distribution
40
35
30
25
20
15
10
5
0
39
20
23
13
7
5
3
11 – 20
21 – 30
31 – 40
41 – 50
51 – 60
61 – 70
71 – 80

TABLE II

Sex Distribution

Sex	No of Cases	Percentage
Male	84	76.3
Female	26	23.6
Total	110	100

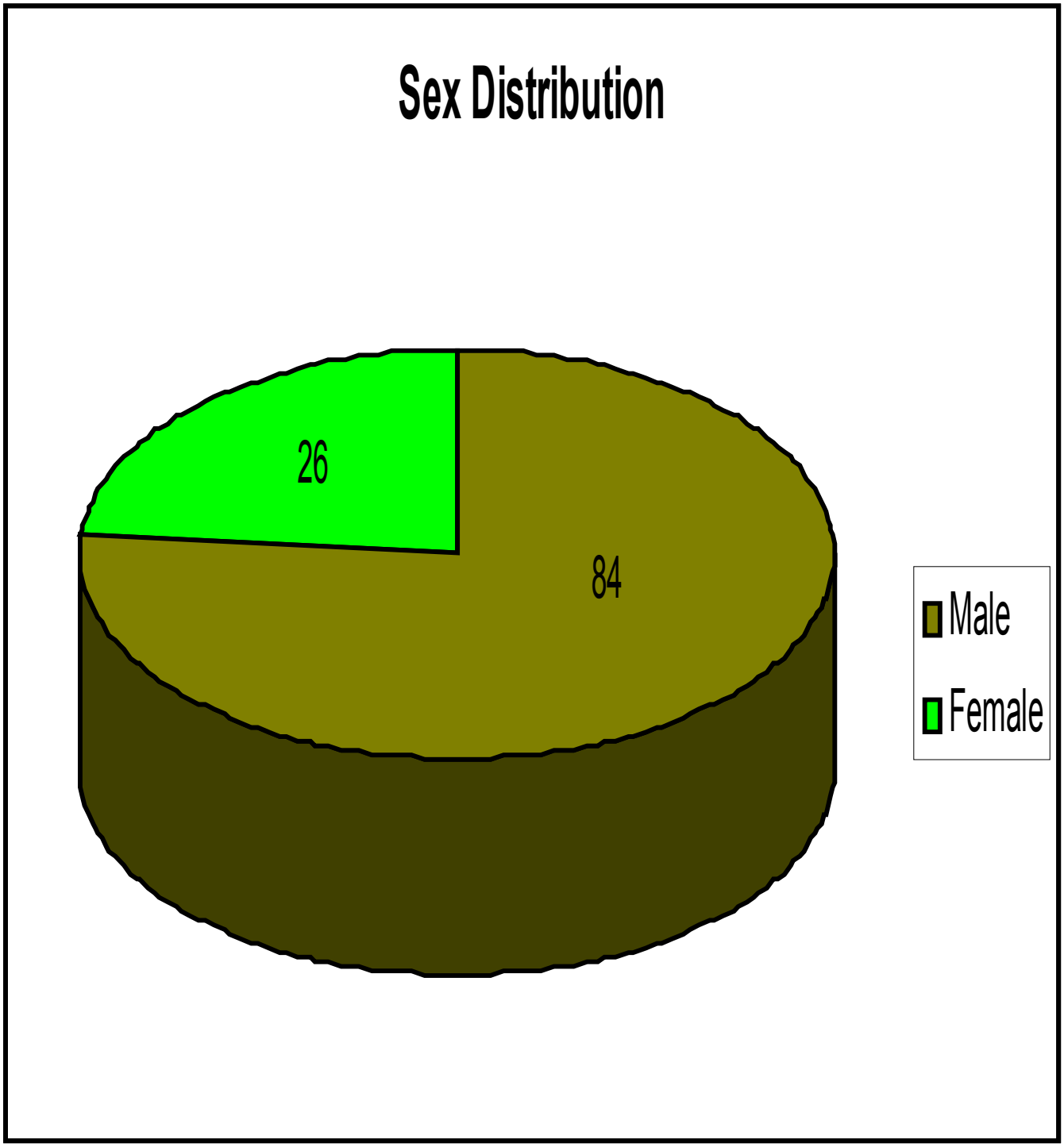

Sex Distribution
26
84
Male
Female

TABLE III

Peripheral Causes

Sl. No	Causes	No of Cases			Percentage
		Male	Female	Total	
1.	CSOM	10	4	14	12.7
2.	Post Operative after Tympanomastoid, Tapes Surgery	4	2	6	5.4
3.	BPPV	16	3	19	17.2
4.	Meniere's Disease	8	3	11	10
5.	Labrinthitis	6	1	7	6.3
6.	Acoustic Neuroma	1	1	2	1.8
7.	Vestibular Neuronitis	14	2	16	14.5

BPPV has the highest incidence - 17.2% followed by

Vestibular Neuronitis - 14.5%

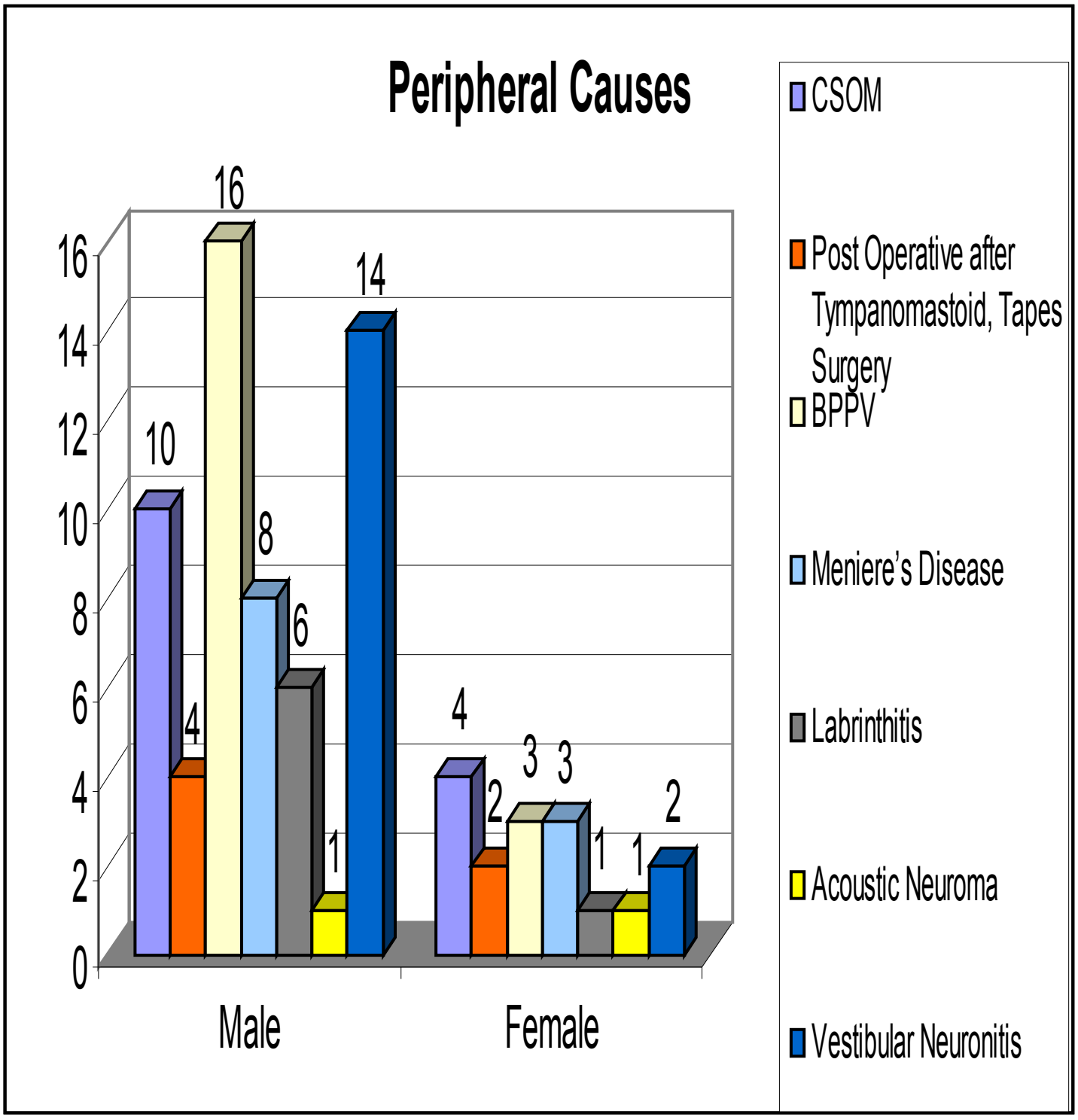

Peripheral Causes
16
14
12
10
8
6
4
2
0
10
4
16
8
6
1
14
4
2
3
3
1
1
2
Male
Female
CSOM
Post Operative after Tympanomastoid, Tapes Surgery
BPPV
Meniere's Disease
Labrinthitis
Acoustic Neuroma
Vestibular Neuronitis

TABLE IV

Central Causes

Sl. No	Causes	No of Cases			Percentage
		Male	Female	Total	
1.	Migrainous Vertigo	4	6	10	9
2.	Vertebro Basilar Insufficiency (VBI)	5	1	6	5.4
3.	Posterior Circulation Stroke	5	0	5	4.5

Migranious Vertigo has the highest incidence of - 9% followed by

Vertebro Basilar Insufficiency (VBI) - 5.4%

Age wise VBI has significant 'P' value <0.001.

Sex wise Migranous vertigo has significant 'P' value <0.005

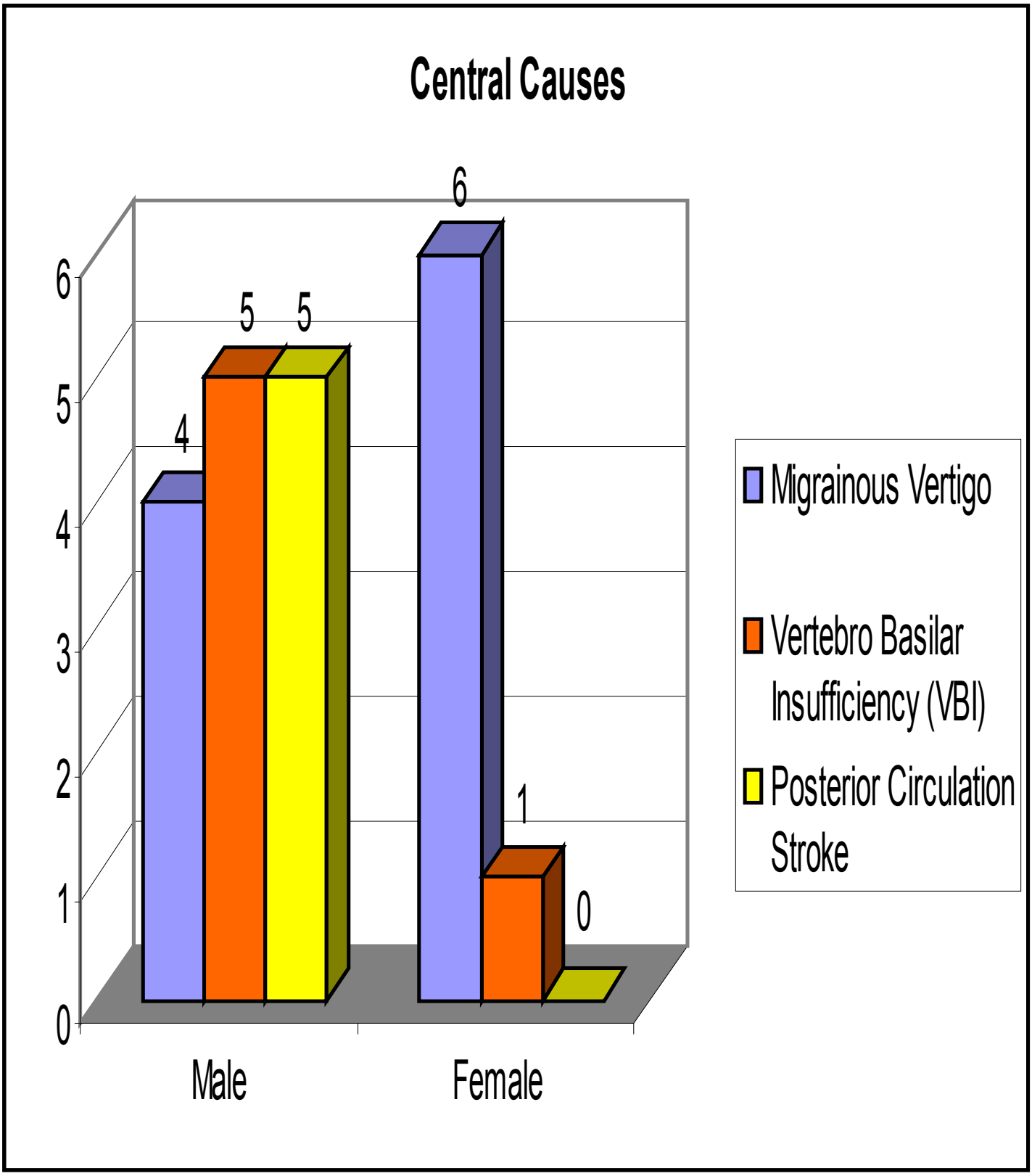

Central Causes
6
5
5
4
1
0
6
5
4
3
2
1
0
Male
Female
Migrainous Vertigo
Vertebro Basilar Insufficiency (VBI)
Posterior Circulation Stroke

TABLE V

Systemic Causes

Sl. No	Causes	No of Cases			Percentage
		Male	Female	Total	
1.	Hypothyroidism	2	1	3	2.7
2.	Diabetes Mellitus	7	2	9	8.1
3.	Drugs – Anti Hypertensive	1	0	1	0.9
4.	Anaemia	1	0	1	0.9

Diabetes Mellitus has the highest incidence of - 8.1%

Systemic Causes

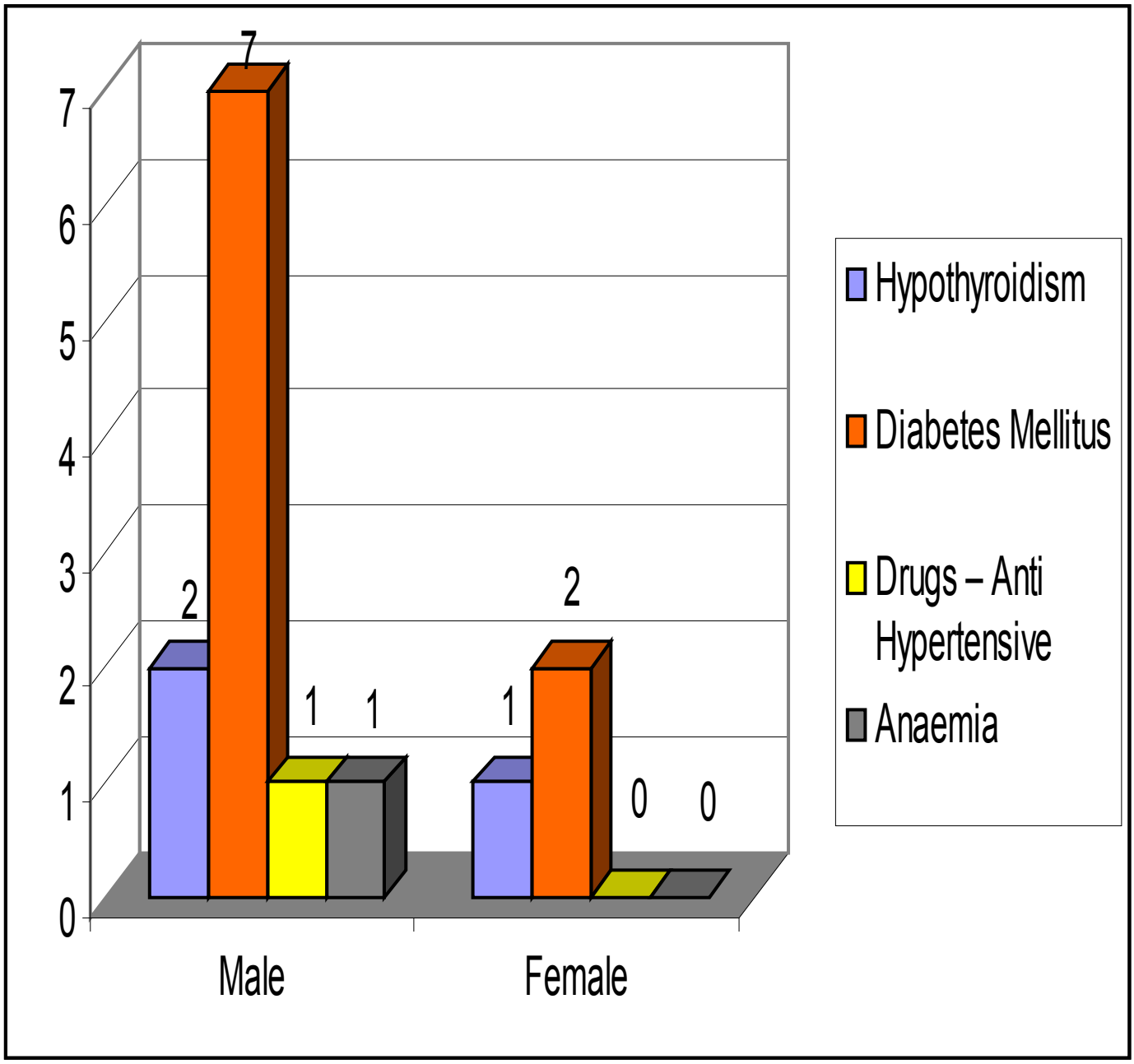

7
6
5
4
3
2
1
0
7
2
1
1
2
1
0
0
Male
Female
Hypothyroidism
Diabetes Mellitus
Drugs – Anti Hypertensive
Anaemia

TABLE VI

Age wise, Sex wise Etiological Spectrum

Age Group	Total No of Persons	Central Causes	Peripheral Causes	Systemic Causes
11 – 20	7	1	6	0
21 – 30	20	3	17	0
31 – 40	39	6	30	3
41 – 50	23	3	17	3
51 – 60	13	4	3	6
61 – 70	5	2	2	1
71 – 80	3	2	0	1

Age wise, Sex wise Etiological Spectrum

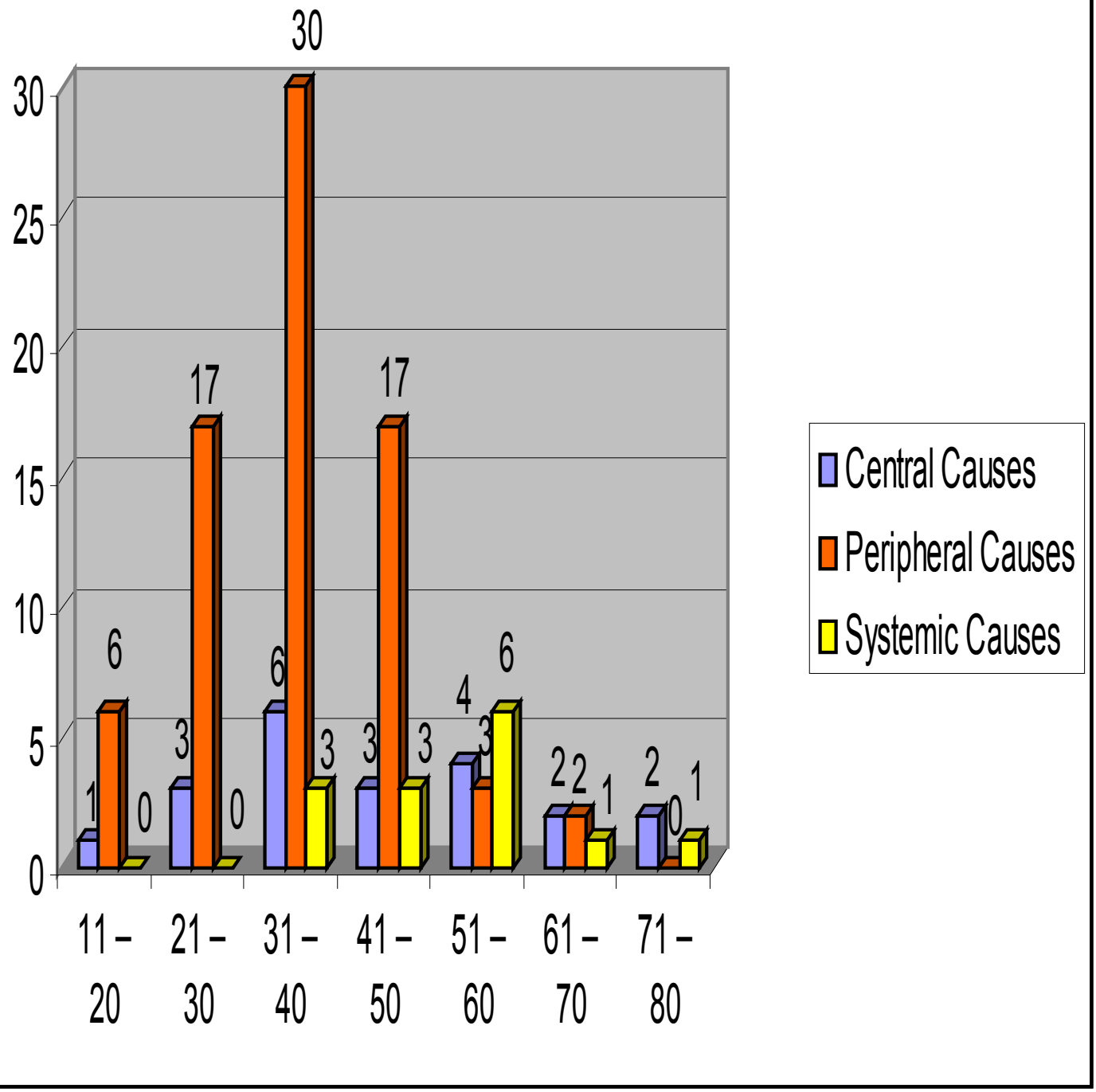


TABLE VII

Causes of Vertigo

Sl. No	Causes	No of Cases			Percentage
		Male	Female	Total	
	Peripheral				
1.	CSOM	10	4	14	12.7
2.	Post Operative after Tympanomastoid, Tapes Surgery	4	2	6	5.4
3.	BPPV	16	3	19	17.2
4.	Meniere's Disease	8	3	11	10
5.	Labrinthitis	6	1	7	6.3
6.	Acoustic Neuroma	1	1	2	1.8
7.	Vestibular Neuronitis	14	2	16	14.5
	Central				
1.	Migrainous Vertigo	4	6	10	9
2.	Vertebro Basilar Insufficiency (VBI)	5	1	6	5.4
3.	Posterior Circulation Stroke	5	0	5	4.5
	Systemic				
1.	Hypothyroidism	2	1	3	2.7
2.	Diabetes Mellitus	7	2	9	8.1
3.	Drugs – Anti Hypertensive	1	0	1	0.9
4.	Anaemia	1	0	1	0.9

TABLE VIII

Bed side Tests for Vertigo

Bed Side Tests	Male	Female	Total
Nystagmus	38	9	47
Fukuda's	28	7	35
Dick's Hallpick's	16	3	19
Head Thrust	30	8	38
Dynamic Visual Acuity	29	5	34
Head Shaking Test	24	6	30
Calorie Test	28	6	34

'P' value for Nystagmus 0.127 (Not Significant)

'P' value for all bedside tests <0.001 which is highly significant.

('P' value <0.05 is significant)

(Positive)

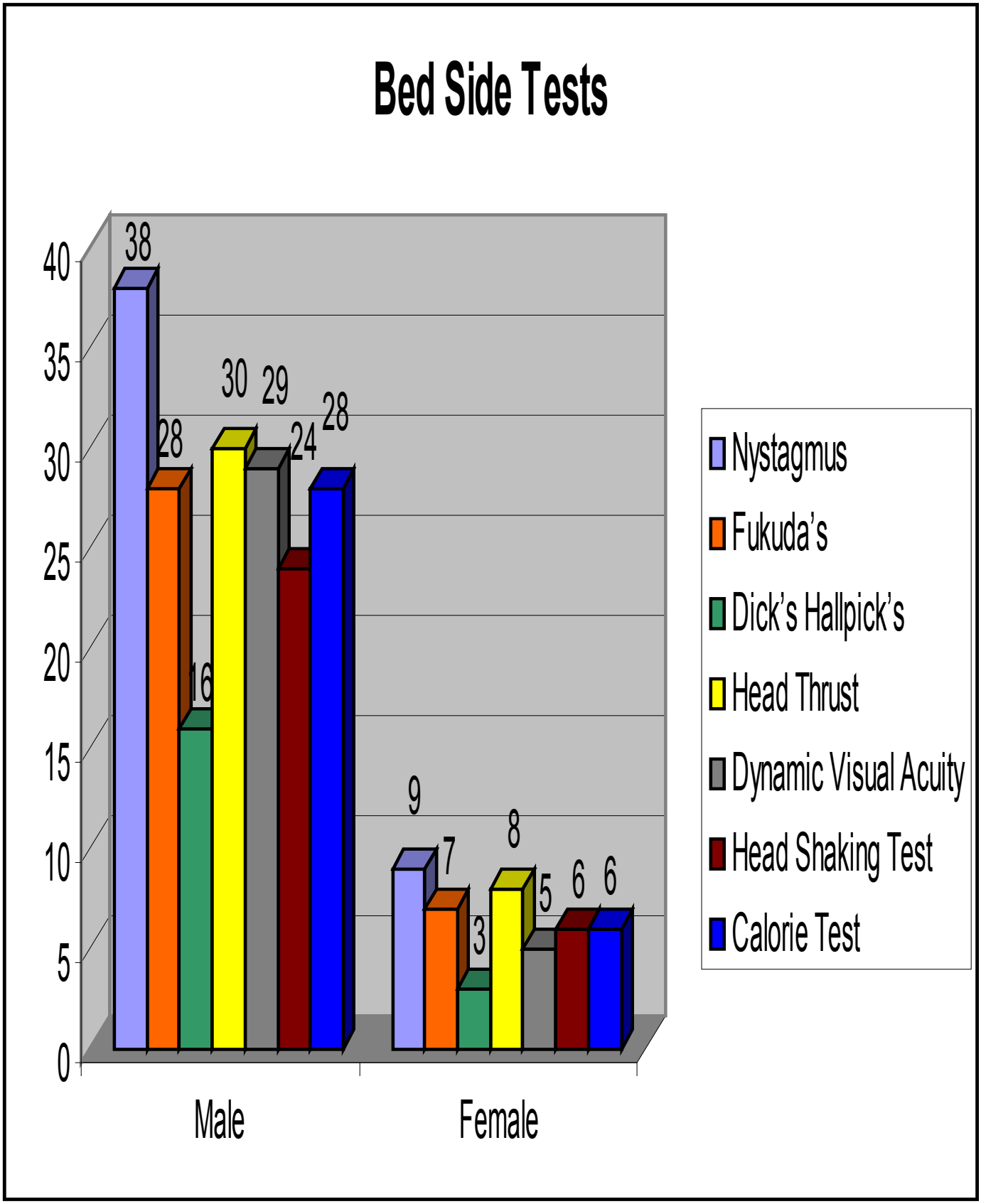

Bed Side Tests
40
35
30
25
20
15
10
5
0
38
28
16
30
29
24
28
9
7
3
8
5
6
6
Male
Female
Nystagmus
Fukuda's
Dick's Hallpick's
Head Thrust
Dynamic Visual Acuity
Head Shaking Test
Calorie Test

TABLE IX

	Fukuda's	Dick Hallpick's	Headthrust	Dynamic VA	Head Shaking	Calorie
CSOM	13	-	4	4	3	-
Post Operative	6	-	3	3	3	-
BPPV	-	19	-	-	1	-
Meniere's	5	1	5	5	4	9
Labrinthitis	7	-	6	5	4	7
Acoustic Neuroma	2	-	1	-	-	2
Vestibular Neuronitis	-	-	16	14	12	16
Migranous Vertigo	-	-	-	-	-	-
VBI	-	-	-	-	-	-
Post Circulation Stroke	2	-	3	3	3	-

TABLE X

Imaging in Vertigo Patient

S.No	Investigation	Male	Female	Total
1	CT Brain	5	1	6
2	MRI Brain	6	1	7
3	Carotid Vertebral Doppler	10	1	11

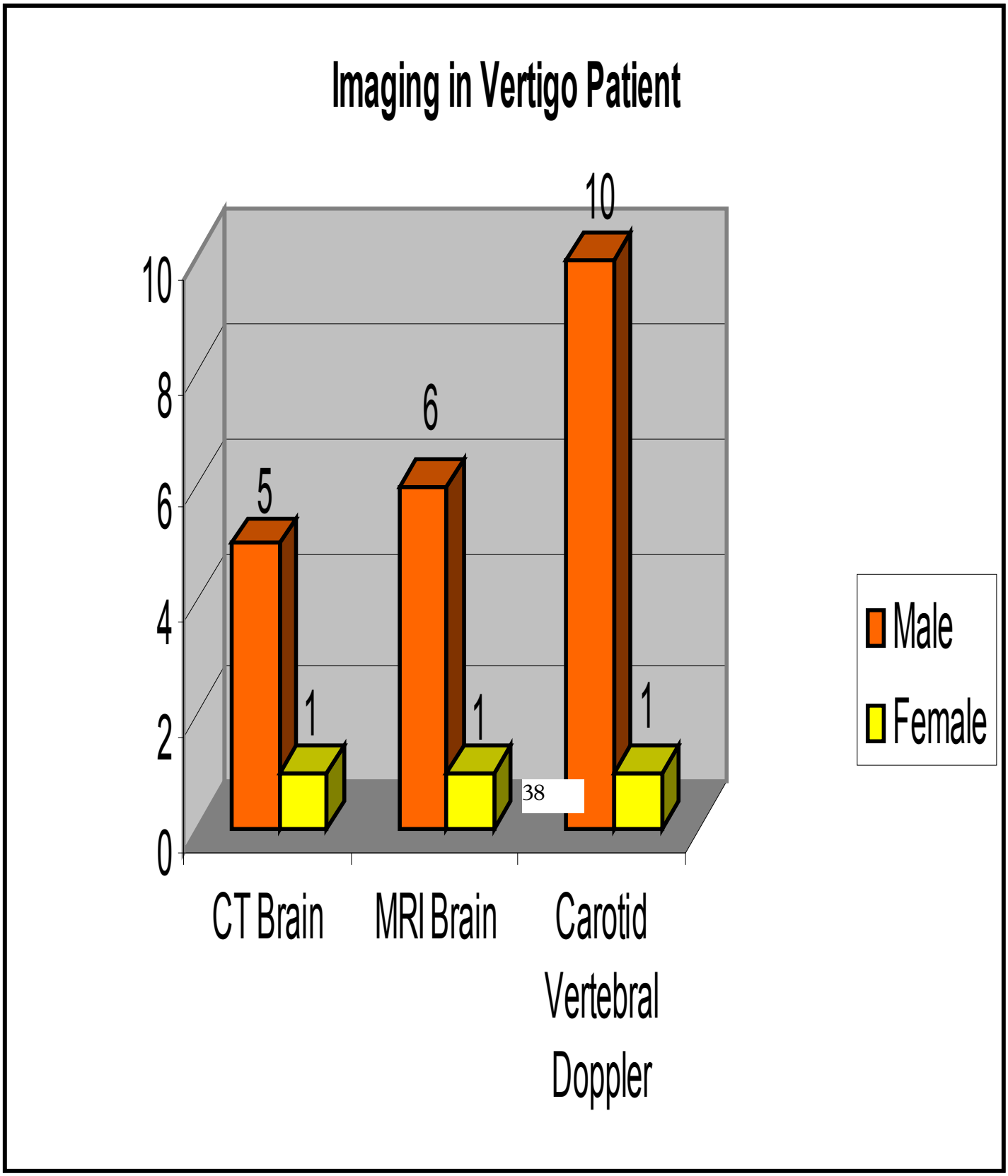


TABLE XI

Associated features with Vertigo

S.No	Associated features with vertigo	Male	Female	Total
1	Hearing Impairment	29	11	40
2	Tinnitus	21	9	30
3	Aural fullness	8	3	11
4	Dysphagia / dysarthria	8	1	9

All associated features have significant ‘P’ value <0.001

Associated features with Vertigo

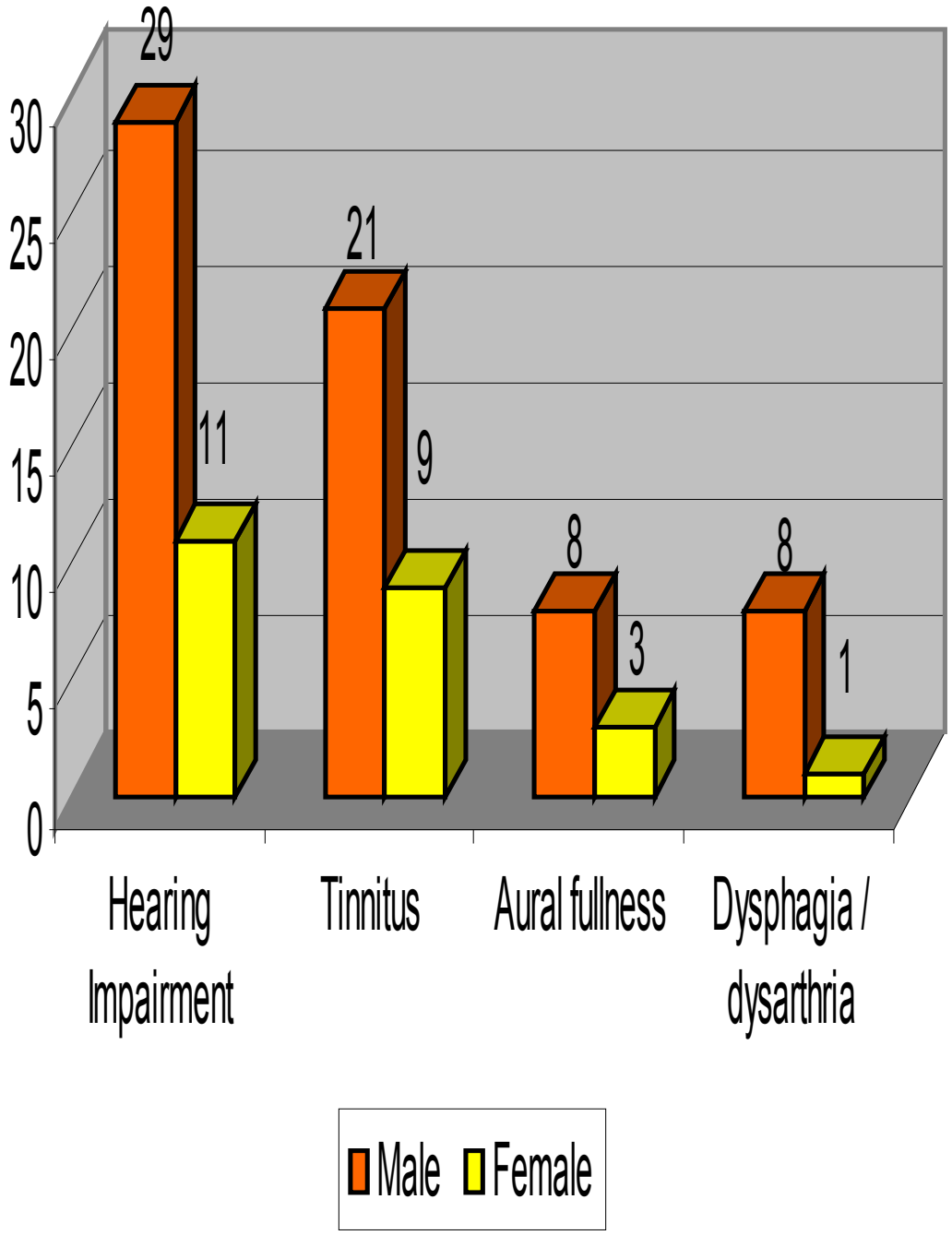
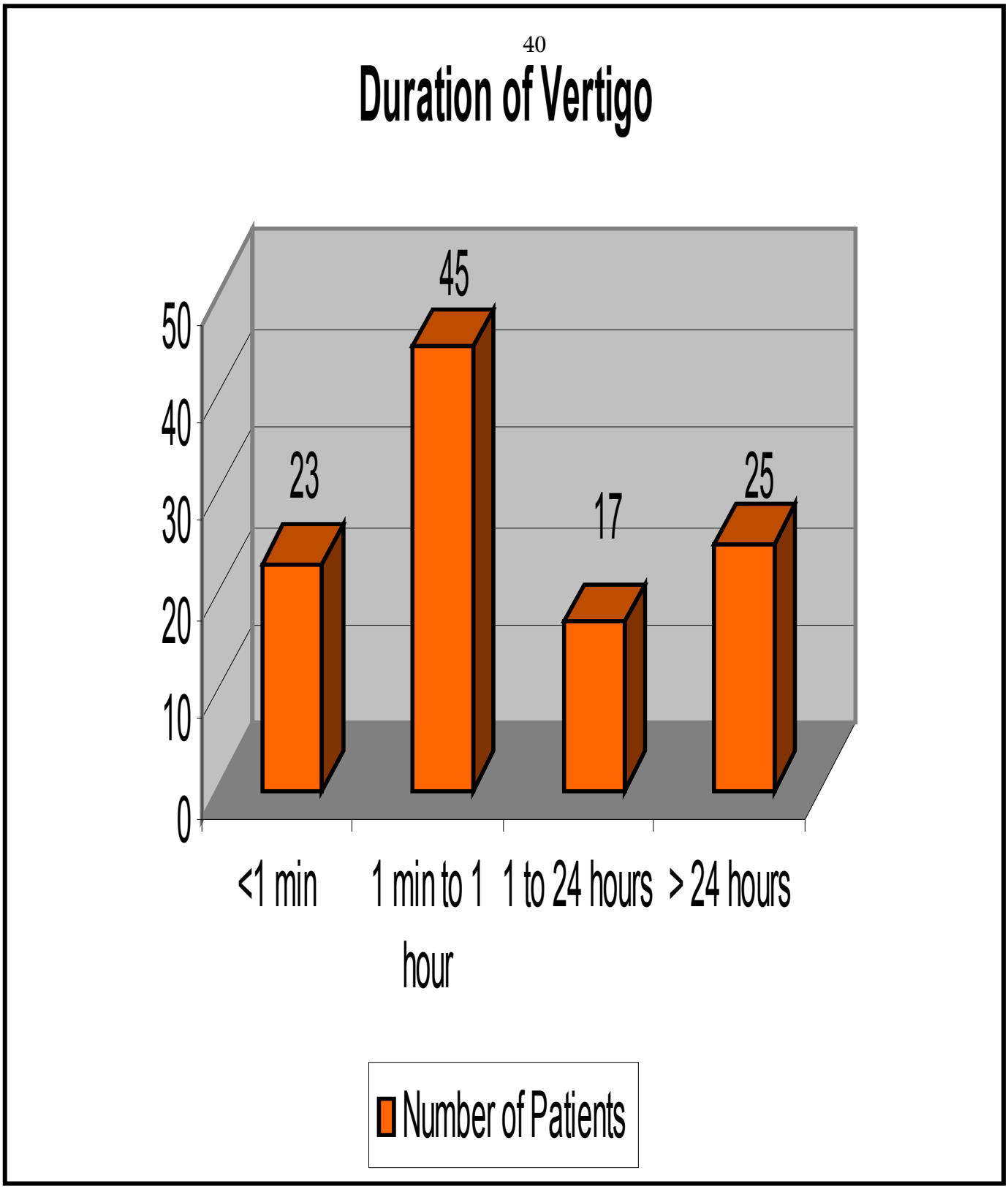


TABLE XII

Duration of Vertigo

-

Duration	<1 min	1 min to 1 hour	1 to 24 hours	> 24 hours
Number of Patients	23	45	17	25


Duration of Vertigo
50
40
30
20
10
0
23
45
17
25
<1 min
1 min to 1 hour
1 to 24 hours
> 24 hours
Number of Patients

DISCUSSION

DISCUSSION

Vertigo is often an untreated symptom and is frequently associated with serious handicap and considerable psychological morbidity. The exact cause often remains elusive. Usually it begins in the fourth decade and attains its highest prevalence in the sixth decade (McNally and Stuart 1949; Nsamba C 1972; Ponniah RD 1977, Bhatia and Deka 1985) (19, 20, 21, 22).

Vertigo disorders of peripheral origin are more commonly found in 3rd and 4th decade of life. The disease is less common above 50 years and is rare below 20 years of age. This study corroborates with the findings of Deka (1985 and Debasish Burman) (23).

In this study the highest incidence is found in 4th decade 39 (35.4%) cases followed by 5th decade 23 (20.9%) cases then 3rd decade 20 (18.1%) cases.

Men appear to be affected more than Women. Men 84 (76.3%) Women 26 (23.6%).

Katsarkas (1994) in a study on dizziness in the elderly reported a prevalence of 63% in women as compared to 37% in men (24). In another population based study on incidence of begin paroxysmal positional vertigo, 64% of affected patients were women (Froehling and Silverstein 1991) (25).

According to Deka (1985), the male and the female ratio of peripheral vertigo 3:2, while Gopal G. S (1991) (28) showed the ratio as 4:1.

In Debasish Burman study males are five folds more affected than females.

BPPV appears to be the commonest disorder (17.2%). Debasish Burman 20% (Montadon 1984, (27) 28%; Deka et al 1985, 17%). Its frequency is probably greater than indicated, because the physicians tend to refer these cases less, being aware of its specificity and food prognosis.

Post operative vertigo found in 6 (5.4%) cases Debasish Burman 7 cases out of 95 cases.

Meniere's disease which is supposed to be the commonest peripheral vestibular disorder (Mawson and Ludman 1979) (28), is less commonly seen in this study 11 (10%) cases. This figure is almost same as studied by Deka (1985) and Debasish Burman (10.5%). The diagnosis presents problems and various centers have their own parameter to diagnose this condition. We diagnosed them on the basis of

Diagnostic criteria of Meniere's Disease.

Labyrinthitis composed of 7 (6.3%) of all patients and vertigo was the predominant complaint in all cases. According to Debasish Burman labyrinthitis was 7.4% of all cases.

Middle ear lesions (CSOM, Post operative cases) comprised of 20 (18.1%) cases in this study. In Debasish Burman study this was 25.2%. It can not be overemphasized that in any vertiginous patient, middle ear disease must be excluded before other avenues of investigations are pursued.

Systemic disorder masquerading as peripheral vertigo is not uncommon. In this study diabetes mellitus 9 (8.1%) cases form the majority. Other findings are hypothyroidism 3 (2.7%) cases. Anti hypertensive induced 1 (0.9%) case. Anaemia induced 1 (0.9%) case.

Acoustic neuroma accounts for about 2 (1.8%) patients as compared to Montadon (1984) 4% and Deka (1985) 10%, Debasish Burman (13.6%)

In this study migranous vertigo in 10 (9%) patients. Vertebro basilar insufficiency 6 (5.4%) and posterior circulation stroke 5 (4.5%).

In a study by Kathleen A. Delaney cerebrovascular disease accounted for 19% of diagnosed causes of vertigo (29).

In one unselected series of 82 patients with migraine headaches, 44 (54%) reported vertigo that was either part of the aura (11/44), accompanied the headache (10/44), or followed the headache (2/44). Other neuro-otologic signs, such as tinnitus and hearing loss, occurred in 15 of 44 and 13 of 44, respectively.

Neuhauser H study showed the prevalence of migranous vertigo was 7% in the dizziness clinic group, and 9% in the migraine clinic group (15).

Nineteen percent of patients with vertebrobasilar insufficiency report at least one episode of isolated vertigo occurring from 1.5 years to two days before the development of multiple symptoms.

In this study vertigo of less than 1 min duration occurs in 23 patients and more than 24 hours occurs in 25 patients. Ronald H. Labuguen showed vertigo of several seconds to a few minutes occurs in Benign paroxysmal positional vertigo; vertigo of Days occurs in Early acute vestibular neuronitis, stroke; migraine (32).

Study by Michael Vonbravern in migranous vertigo in 2005 showed out of 20 patients, 6 patients showed duration of less than 6 hours, 8 patients showed duration of 24 hours to 1 week, 4 patients showed duration of more than 1 week (30).

In this study hearing impairment occurs in 40 patients, tinnitus occurs in 30 patients, aural fullness occurs in 11 patients, dysphagia and dysarthria occurs in 9 patients. Study by Kathleen A. Delaney showed hearing loss 14%, tinnitus 10%, dysphagia 10%.

In this study CT brain showed lateral medullary infarct in 4 patients. MRI brain showed lateral medullar infarct in 5 patients. In a study by A Barth PICA infarct manifested as acute vertigo. Total No of study patients 34. 16 patients had PICA infarct and all the 16 patients had vertigo (31).

While doing fukuda's test 9 patients moving forward 3-4 steps instead of turning to one side.

All the 19 cases of BPPV fulfill all the 4 diagnostic criteria for BPPV and Dick Hallpick's test was positive in all the 19 BPPV patients.

Among the 10 patients of Migranous vertigo 8 patients fulfill the criteria for definite Migranous vertigo 2 patients were probable Migranous vertigo.

In this study the bedside tests were not useful to identify Migranous vertigo.

Nystagmus was presented 47 patients out of this 36 patients had features of peripheral nystagmus, 11 patients had features of central nystagmus.

The Bedside tests used in this study were very much useful for vertigo with the

significant ‘P’ value of < 0.001.

Various bedside tests applied in this study among these most of the tests positive in both central and peripheral vertigo.

Two tests (Calorie, Dick Hall pick’s) were specific for peripheral vertigo.

Head thrust test is the most significant test in this study in this 46.66% positive for peripheral vertigo and 14.28% positive for central vertigo, so more than half (53.34%) of peripheral vertigo cases it is negative.

Calorie tests positive only in peripheral vertigo (45.33%) but in more than half (54.77%) of patients of peripheral vertigo cases it is negative. Even though calorie test is specific for peripheral vertigo the absence of this test will not exclude the peripheral vertigo.

Dick Hal pick’s test is very specific for BPPV (100% positive) and it is not positive in central cases.

Dynamic visual acuity is positive in 41.33% of peripheral cases and 14.28% of central cases of vertigo.

Fukuda’s test is positive in 44% of peripheral cases and 9.52% of central cases of

vertigo.

Head shaking test is positive in 36% of peripheral cases and 14.28% of central cases of vertigo.

The Head thrust test, Dynamic Visual Acuity, Fukuda's test and Head Shaking test are positive nearly 40% cases of peripheral vertigo but was not specific for a particular Aetiopathology.

An analysis of the various bedside tests employed in the assessment of vertigo revealed that most often the tests are positive in patients with peripheral causes rather than central.

All the tests were not positive in a given patient but at least one of these tests was abnormal in patients with a peripheral vertigo.

In patients in whom the test was positive with suspected central cause, all of them had posterior circulation stroke which could have involved the peripheral apparatus as well.

Those with peripheral causes in whom bedside tests were positive, a pattern or correlation of a test with a particular Aetiopathology was observed.

The Dick Halpike's test was positive in all patients with BPPV and was found to be specific for this condition and not seen with other peripheral or central causes.

The Calorie test was positive in patients with peripheral causes of vertigo only but was positive in only less than half of the number of patients, mostly in patients with labrynthine (45.5%) or vestibular nerve involvement.

SUMMARY

SUMMARY

Total number of vertigo patients- 110

31 to 40 years of age was the most common age group affected in the study. 39 (35.4%.)

Total number of male patients 84 (76.3%)

Total number of female patients 26(23.6%)

BPPV was the commonest peripheral cause of vertigo 19 (17.2%)

Migranous Vertigo was the commonest central cause of Vertigo 10(9%)

Diabetes mellitus was the commonest systemic cause of vertigo.9(8.1%)

Overall BPPV was the commonest cause 19 (17.2%) in this study.

Vestibular neuronitis 16 (14.5%) was the second common cause of vertigo

6 (5.4%) patients had vertebro basilar insufficiency

5 (4.5%) patients had posterior circulation stroke

47 patients had nystagmus

Among the bedside test head thrust test was most significant, and 38 patients showed positive results with head thrust test.

Dick Halpick's, Calorie test positive only in peripheral vertigo.

CT brain showed lateral medullary infarct in 4 patients and acoustic neuroma in 2 patients.

MRI brain showed lateral medullary infarct in 5 patients and acoustic neuroma in 2 patients.

Vertigo duration of less than 1 min occurs in 23 patients more than 24 hours occurs in 25 patients.

Vertigo associated with hearing important in 40 patients. Tinnitus in 30 patients dysphagia and dysarthria in 9 patients.

CONCLUSION

CONCLUSION

Benign paroxysmal positional vertigo (BPPV) was the commonest cause of vertigo in this study.

31 to 40 years had the highest incidence of vertigo in this study.

Migranous vertigo was the common central cause of vertigo.

Head thrust test was the most reliable bedside test.

Dick Halpick's and calorie test positive only in peripheral vertigo.

All the bedside tests used in this study were very much significant for vertigo with the 'P' value of <0.001.

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PROFORMA

PROFORMA

Name Age Sex MIN No.

Vertigo

Duration	frequency	effect of head position/movement
Hearing Impairment	tinnitus	auralfullness
Nauesa,Vomitting	visual blurring	

Headache

Ear Discharge.

Ear surgery

Drugs

Joint pain,swelling,rash

Chest pain/Palpitation

Anxiety/Hyperventillation

Double vision

Dysphagia/dysarthria

Dimness of vision

Limb weakness, numbness

FamilyH/O

Diabetes

Neck pain/restriction of neck movements

EXAMINATION

PR

BP	Supine	Standing	Carotids
Vestibuloocular reflex	Pursuit	Nystagmus	OKN
Saccades	Vergence	Visual acuity	Fundus
EOM			

Lower Cranial nerves

SMS -- Coordination Sensory
Involuntary Movements Gait

Cerebellar signs	Rombergs	Tandomrombergs
Fukuda's	Pastpointing	Dick's Hallpick's
Head Thrust-Test	Dynamic visual acuity	
Head shaking test	Calorie Test	

INVESTIGATIONS

Audiogram

CT / MRI Brain

X-Ray Neck – AP, Lateral view

Blood sugar --Fasting, Post prandial

Lipid Profile
Carotid Vertebral Doppler